Exhibit F

Fetal Alcohol Syndrome Article
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Fetal alcohol syndrome

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(Redirected from Fetal Alcohol Effects)

Fetal alcohol syndrome (FAS) is a disorder of permanent birth defects that occurs in the offspring of women who drink alcohol during pregnancy. It is unknown whether amount, frequency or timing of alcohol consumption during pregnancy causes a difference in degree of damage done to the fetus. Thus, although prenatal alcohol exposure does not automatically result in FAS, the current recommendation of the US Surgeon General is not to drink at all during pregnancy.[1]

Fetal Alcohol Syndrome (FAS)	
Classification and external resources	
ICD-10	Q86.0. (http://www.who.int/classifications/apps/icd/icd10online/?gq86.0.htm+q86.0)
ICD-9	760.71 (http://www.icd9data.com/getICD9Code.ashx?icd9=760.71)
DiseasesDB	32957 (http://www.diseasesdatabase.com/ddb32957.htm)
MedlinePlus	000911 (http://www.nlm.nih.gov/medlineplus/ency/article/000911.htm)
eMedicine	ped/767 (http://www.emedicine.com/ped/topic767.htm)
MeSH	D005310 (http://www.nlm.nih.gov/cgi/mesh/2008/MB_cgi?field=uid&term=D005310)

Alcohol and Health
Short-term effects of alcohol
Long-term effects of alcohol
Alcohol and cardiovascular disease
Alcoholic liver disease
Alcoholic hepatitis
Alcohol and cancer
Alcohol and weight
Fetal alcohol syndrome
Fetal Alcohol Spectrum Disorder
Alcoholism
Blackout (alcohol-related amnesia)
Wernicke-Korsakoff syndrome
Recommended maximum intake

Alcohol crosses the placental barrier and can stunt fetal growth or weight, create distinctive facial stigmata, damage neurons and brain structures, and cause other physical, mental, or behavioral problems.[2][3][4] Surveys found that in the United States, 10-15% of pregnant women admit to having recently used alcohol, and up to 30% use alcohol at some point during pregnancy.[5][6][7] The main effect of FAS is permanent central nervous system damage, especially to the brain. Developing brain cells and structures are underdeveloped or malformed by prenatal alcohol exposure, often creating an array of primary cognitive and functional disabilities (including poor memory, attention deficits, impulsive behavior, and poor cause-effect reasoning) as well as secondary disabilities (for example, mental health problems, and drug addiction).[4][8] The risk of brain damage exists during each trimester, since the fetal brain develops throughout the entire pregnancy.[9]

Fetal alcohol exposure is the leading known cause of mental retardation in the Western world.[10] In the United States the FAS prevalence rate is estimated to be between 0.2 and 2.0 cases per 1,000 live births, comparable to or higher than other developmental disabilities such as Down syndrome or spina bifida.[11] The lifetime medical and social costs of each child with FAS are estimated to be as high as US$800,000.[12]

Contents

History

Historical references

Anecdotal accounts of prohibitions against maternal alcohol use from biblical, ancient Greek, and ancient Roman sources imply a historical awareness of links between maternal alcohol use and negative child outcomes.[13]

The earliest known observation of possible links between maternal alcohol use and fetal damage was made in 1899 by Dr. William Sullivan, a Liverpool prison physician who noted higher rates of stillbirth for 120 alcoholic female prisoners than their sober female relatives; he suggested the causal agent to be alcohol use.[14] This contradicted the predominating belief at the time that heredity caused mental retardation, poverty, and criminal behavior, which contemporary studies on the subjects usually concluded.[4] A case study by Henry H. Goddard of the Kallikak family — popular in the early 1900s — represents this earlier perspective,[15] though later researchers have suggested that the Kallikaks almost certainly had FAS.[16] General studies and discussions on alcoholism throughout the mid-1900s were typically based on a heredity argument.[17]

Prior to fetal alcohol syndrome being specifically identified and named in 1973, a few studies had noted differences between the children of mothers who used alcohol during pregnancy or breast-feeding and those who did not, but identified alcohol use as a possible contributing factor rather than heredity.[4]

Recognition as a syndrome

Fetal Alcohol Syndrome was named in 1973 by two dysmorphologists, Drs. Kenneth Lyons Jones and David Weyhe Smith of the University of Washington Medical School in Seattle, United States. They identified a pattern of "craniofacial, limb, and cardiovascular defects associated with prenatal onset growth deficiency and developmental delay" in eight unrelated children of three ethnic groups, all born to mothers who were alcoholics.[18] The pattern of malformations indicated that the damage was prenatal. News of the discovery shocked some, while others were skeptical of the findings.[19]

Dr. Paul Lemoine of Nantes, France had already published a study in a French medical journal in 1968 about children with distinctive features whose mothers were alcoholics,[3] and in the U.S., Christy Ulleland and colleagues at the University of Washington Medical School[2] had conducted an 18-month study in 1968-1969 documenting the risk of maternal alcohol consumption among the offspring of 11 alcoholic mothers. The Washington and Nantes findings were confirmed by a research group in Gothenburg, Sweden in 1979.[20] Researchers in France, Sweden, and the United States were struck by how similar these children looked, though they were not related, and how they behaved in the same unfocused and hyperactive manner.[20]

Within four years of the Washington discovery, animal studies, including non-human primate studies carried out at the University of Washington Primate Center by Dr. Sterling Clarren, had confirmed that alcohol was a teratogen. By 1978, 245 cases of FAS had been reported by medical researchers, and the syndrome began to be described as the most frequent known cause of mental retardation.

While many syndromes are eponymous, i.e. named after the physician first reporting the association of symptoms, Dr. Smith named FAS after the causal agent of the symptoms.[21] He reasoned that doing so

would encourage prevention, believing that if people knew maternal alcohol consumption caused the syndrome, then abstinence during pregnancy would follow from patient education and public awareness.[21] Nobody was aware of the full range of possible birth defects from FAS or its prevalence rate at that time,[21] but admission of alcohol use during pregnancy can feel stigmatizing to birth mothers and complicate diagnostic efforts[11] of a syndrome with its preventable cause in the name.

Over time, as subsequent research and clinical experience suggested that a range of effects (including physical, behavioral, and cognitive) could arise from prenatal alcohol exposure, the term Fetal Alcohol Spectrum Disorder (FASD) was developed to include FAS as well as other conditions resulting from prenatal alcohol exposure.[21] Currently, FAS[18][22][23] is the only expression of prenatal alcohol exposure defined by the International Statistical Classification of Diseases and Related Health Problems and assigned ICD-9 and ICD-10 diagnoses.

Diagnosis

Several diagnostic systems have been developed in North America:

- The Institute of Medicine's guidelines for FAS, the first system to standardize diagnoses of individuals with prenatal alcohol exposure,[23]
- The University of Washington's "The 4-Digit Diagnostic Code," which ranks the four key features of FASD on a Likert scale of one to four and yields 256 descriptive codes that can be categorized into 22 distinct clinical categories, ranging from FAS to no findings,).[24]
- The Centers for Disease Control's "Fetal Alcohol Syndrome: Guidelines for Referral and Diagnosis," which established general consensus on the diagnosis FAS in the U.S. but deferred addressing other FASD conditions,[11] and
- Canadian guidelines for FASD diagnosis, which established criteria for diagnosing FASD in Canada and harmonized most differences between the IOM and University of Washington's systems.[25]

Fetal alcohol syndrome is the only expression of FASD that has garnered consensus among experts to become an official ICD-9 and ICD-10 diagnosis. To make this diagnosis (or determine any FASD condition), a multi-disciplinary evaluation is necessary to assess each of the four key features for assessment. Generally, a trained physician will determine growth deficiency and FAS facial features. While a qualified physician may also assess central nervous system structural abnormalities and/or neurological problems, usually central nervous system damage is determined through psychological, speech-language, and occupational therapy assessments. Prenatal alcohol exposure risk may be assessed by a qualified physician, psychologist, social worker, or chemical health counselor. These professionals work together as a team to assess and interpret data of each key feature for assessment and develop an integrative, multi-disciplinary report to diagnose FAS (or other FASD conditions) in an individual.

The following criteria must be fully met for an FAS diagnosis:[24][23][11][25]

1. Growth deficiency — Prenatal or postnatal height or weight (or both) at or below the 10th percentile[26]
2. FAS facial features — All three FAS facial features present[27]
3. Central nervous system damage — Clinically significant structural, neurological, *or* functional

impairment
4. Prenatal alcohol exposure — Confirmed or Unknown prenatal alcohol exposure

Differential diagnosis

The CDC reviewed nine syndromes that have overlapping features with FAS; however, none of these syndromes include all three FAS facial features, and none are the result of prenatal alcohol exposure:[11]

- Aarskog syndrome
- Williams syndrome
- Noonan's syndrome
- Dubowitz syndrome
- Brachman-DeLange syndrome
- Toluene syndrome
- Fetal hydantoin syndrome
- Fetal valproate syndrome
- Maternal PKU fetal effects

Signs and symptoms

Growth deficiency

Growth deficiency is defined as significantly below average height, weight or both due to prenatal alcohol exposure, and can be assessed at any point in the lifespan. Growth measurements must be adjusted for parental height, gestational age (for a premature infant), and other postnatal insults (e.g., poor nutrition), although birth height and weight are the preferred measurements.[24] Deficiencies are documented when height or weight falls at or below the 10th percentile of standardized growth charts appropriate to the patient's population.[26]

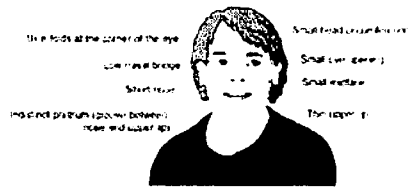
The CDC and Canadian guidelines use the 10th percentile as a cut-off to determine growth deficiency.[11][25] The "4-Digit Diagnostic Code" allows for mid-range gradations in growth deficiency (between the 3rd and 10th percentiles) and severe growth deficiency at or below the 3rd percentile.[24] Growth deficiency (at severe, moderate, or mild levels) contributes to diagnoses of FAS and PFAS (Partial Fetal Alcohol Syndrome), but not ARND (Alcohol-Related Neurodevelopmental Disorder) or static encephalopathy.

Growth deficiency is ranked as follows by the "4-Digit Diagnostic Code:"[24]

- Severe — Height and weight at or below the 3rd percentile.
- Moderate — Either height or weight at or below the 3rd percentile, but not both.
- Mild — Both height and weight between the 3rd and 10th percentiles.
- None — Height and weight both above the 10th percentile.

Facial features

Several characteristic craniofacial abnormalities are visible in individuals with FAS.[28] The presence of FAS facial features indicates brain damage, though brain damage may also exist in their absence. FAS facial features (and most other visible, but non-diagnostic, deformities) are believed to be caused mainly during the 10th and 20th week of gestation.[29]



Facial characteristics of a child with FAS

Refinements in diagnostic criteria since 1975 have yielded three distinctive and diagnostically significant facial features known to result from prenatal alcohol exposure and distinguishes FAS from other disorders with partially overlapping characteristics.[30][31] The three FAS facial features are:

- A smooth philtrum — The divot or groove between the nose and upper lip flattens with increased prenatal alcohol exposure.
- Thin vermilion — The upper lip thins with increased prenatal alcohol exposure.
- Small palpebral fissures — Eye width decreases with increased prenatal alcohol exposure.

Measurement of FAS facial features uses criteria developed by the University of Washington. The lip and philtrum are measured by a trained physician with the Lip-Philtrum Guide,[32] a 5-point Likert Scale with representative photographs of lip and philtrum combinations ranging from normal (ranked 1) to severe (ranked 5). Palpebral fissure length (PFL) is measured in millimeters with either calipers or a clear ruler and then compared to a PFL growth chart, also developed by the University of Washington.[27]

Ranking FAS facial features is complicated because the three separate facial features can be affected independently by prenatal alcohol. A summary of the criteria follows:[24][33]

- Severe — All three facial features ranked independently as severe (lip ranked at 4 or 5, philtrum ranked at 4 or 5, and PFL less than or equal to two standard deviations below average).
- Moderate — Two facial features ranked as severe and one feature ranked as moderate (lip *or* philtrum ranked at 3, *or* PFL between one and two standard deviations below average).
- Mild — A mild ranking of FAS facial features covers a broad range of facial feature combinations:
  - Two facial features ranked severe and one ranked within normal limits,
  - One facial feature ranked severe and two ranked moderate, or
  - One facial feature ranked severe, one ranked moderate and one ranked within normal limits.
- None — All three facial features ranked within normal limits.

These distinctive facial features in a patient do strongly correlate to brain damage. Sterling Clarren of the University of Washington's Fetal Alcohol and Drug Unit told a conference in 2002:

> I have never seen anybody with this whole face who doesn't have some brain damage. In fact in studies, as the face is more FAS-like, the brain is more likely to be abnormal. The only face that you would want to counsel people or predict the future about is the full FAS face. But the risk of brain damage increases as the eyes get smaller, as the philtrum gets flatter, and the lip gets thinner. The risk goes up but not the diagnosis.
>
> At one-month gestation, the top end of your body is a brain, and at the very front end of that early brain, there is tissue that has been brain tissue. It stops being brain and gets ready to be your face ...

> Your eyeball is also brain tissue. It's an extension of the second part of the brain. It started as brain and "popped out." So if you are going to look at parts of the brain from alcohol damage, or any kind of damage during pregnancy, eye malformations and midline facial malformations are going to be very actively related to the brain across syndromes ... and they certainly are with FAS.[34]

Central nervous system damage

Central nervous system (CNS) damage is the primary feature of any FASD diagnosis. Prenatal alcohol exposure, a teratogen, can damage the brain across a continuum of gross to subtle impairments, depending on the amount, timing, and frequency of the exposure as well as genetic predispositions of the fetus and mother.[23][35] While functional abnormalities are the behavioral and cognitive expressions of the FAS disability, CNS damage can be assessed in three areas: structural, neurological, and functional impairments.

All four diagnostic systems allow for assessment of CNS damage in these areas, but criteria vary. The IOM system requires structural or neurological impairment for a diagnosis of FAS.[23] The "4-Digit Diagnostic Code" and CDC guidelines state that functional anomalies must measure at two standard deviations or worse in three or more functional domains for a diagnoses of FAS.[24][11] The "4-Digit Diagnostic Code" further elaborates the degree of CNS damage according to four ranks:

- Definite — Structural impairments or neurological impairments for FAS or static encephalopathy.
- Probable — Significant dysfunction of two standard deviations or worse in three or more functional domains.
- Possible — Mild to moderate dysfunction of two standard deviations or worse in one or two functional domains *or* by judgment of the clinical evaluation team that CNS damage cannot be dismissed.
- Unlikely — No evidence of CNS damage.

Structural

Structural abnormalities of the brain are observable, physical damage to the brain or brain structures caused by prenatal alcohol exposure. Structural impairments may include microcephaly (small head size) of two or more standard deviations below the average, or other abnormalities in brain structure (e.g., agenesis of the corpus callosum, cerebellar hypoplasia).[23]

Microcephaly is determined by comparing head circumference (often called occipitofrontal circumference, or OFC) to appropriate OFC growth charts.[26] Other structural impairments must be observed through medical imaging techniques by a trained physician. Because imaging procedures are expensive and relatively inaccessible to most patients, diagnosis of FAS is not frequently made via structural impairments, except for microcephaly.

Evidence of a CNS structural impairment due to prenatal alcohol exposure will result in a diagnosis of FAS, and neurological and functional impairments are highly likely.[23][24][11][25]

During the first trimester of pregnancy, alcohol interferes with the migration and organization of brain cells, which can create structural deformities or deficits within the brain.[36] During the third trimester, damage can be caused to the hippocampus, which plays a role in memory, learning, emotion, and

encoding visual and auditory information, all of which can create neurological and functional CNS impairments as well.[37]

As of 2002, there were 25 reports of autopsies on infants known to have FAS. The first was in 1973, on an infant who died shortly after birth.[13] The examination revealed extensive brain damage, including microcephaly, migration anomalies, callosal dysgenesis, and a massive neuroglial, leptomeningeal heterotopia covering the left hemisphere.[38]

In 1977, Dr. Clarren described a second infant whose mother was a binge drinker. The infant died ten days after birth. The autopsy showed severe hydrocephalus, abnormal neuronal migration, and a small corpus callosum (which connects the two brain hemispheres) and cerebellum.[38] FAS has also been linked to brainstem and cerebellar changes, agenesis of the corpus callosum and anterior commissure, neuronal migration errors, absent olfactory bulbs, meningomyelocele, and porencephaly.[38]

Neurological

When structural impairments are not observable or do not exist, neurological impairments are assessed. In the context of FAS, neurological impairments are caused by prenatal alcohol exposure which causes general neurological damage to the central nervous system (CNS), the peripheral nervous system, or the autonomic nervous system. A determination of a neurological problem must be made by a trained physician, and must not be due to a postnatal insult, such as a high fever, concussion, traumatic brain injury, etc.

All four diagnostic systems show virtual agreement on their criteria for CNS damage at the neurological level, and evidence of a CNS neurological impairment due to prenatal alcohol exposure will result in a diagnosis of FAS, and functional impairments are highly likely.[23][24][11][25]

Neurological problems are expressed as either hard signs, or diagnosable disorders, such as epilepsy or other seizure disorders, or soft signs. Soft signs are broader, nonspecific neurological impairments, or symptoms, such as impaired fine motor skills, neurosensory hearing loss, poor gait, clumsiness, poor eye-hand coordination, or sensory integration dysfunction. Many soft signs have norm-referenced criteria, while others are determined through clinical judgment.

Functional

When structural or neurological impairments are not observed, all four diagnostic systems allow CNS damage due to prenatal alcohol exposure to be assessed in terms of functional impairments.[23][24][11][25] Functional impairments are deficits, problems, delays, or abnormalities due to prenatal alcohol exposure (rather than hereditary causes or postnatal insults) in observable and measurable domains related to daily functioning, often referred to as developmental disabilities. There is no consensus on a specific pattern of functional impairments due to prenatal alcohol exposure[23] and only CDC guidelines label developmental delays as such,[11] so criteria vary somewhat across diagnostic systems.

The four diagnostic systems list various CNS domains that can qualify for functional impairment that can determine an FAS diagnosis:

- Evidence of a complex pattern of behavior or cognitive abnormalities inconsistent with developmental level in the following CNS domains — sufficient for a PFAS or ARND diagnosis using IOM guidelines[23]
  - Learning disabilities, academic achievement, impulse control, social perception, communication, abstraction, math skills, memory, attention, judgment
- Performance at two or more standard deviations on standardized testing in three or more of the following CNS domains — sufficient for an FAS, PFAS or static encephalopathy diagnosis using 4-Digit Diagnostic Code[24]
  - Executive functioning, memory, cognition, social/adaptive skills, academic achievement, language, motor skills, attention, activity level
- General cognitive deficits (e.g., IQ) at or below the 3rd percentile on standardized testing — sufficient for an FAS diagnosis using CDC guidelines[11]
- Performance at or below the 16th percentile on standardized testing in three or more of the following CNS domains — sufficient for an FAS diagnosis using CDC guidelines[11]
  - Cognition, executive functioning, motor functioning, attention and hyperactive problems, social skills, sensory problems, social communication, memory, difficulties responding to common parenting practices
- Performance at two or more standard deviations on standardized testing in three or more of the following CNS domains — sufficient for an FAS diagnosis using Canadian guidelines
  - Cognition, communication, academic achievement, memory, executive functioning, adaptive behavior, social skills, social communication

Prenatal alcohol exposure

Prenatal alcohol exposure is determined by interview of the biological mother or other family members knowledgeable of the mother's alcohol use during the pregnancy (if available), prenatal health records (if available), and review of available birth records, court records (if applicable), chemical dependency treatment records (if applicable), or other reliable sources.

Exposure level is assessed as Confirmed Exposure, Unknown Exposure, and Confirmed Absence of Exposure by the IOM, CDC and Canadian diagnostic systems. The "4-Digit Diagnostic Code" further distinguishes confirmed exposure as High Risk and Some Risk:

- High Risk — Confirmed use of alcohol during pregnancy known to be at high blood alcohol levels (100mg/dL or greater) delivered at least weekly in early pregnancy.
- Some Risk — Confirmed use of alcohol during pregnancy with use less than High Risk or unknown usage patterns.
- Unknown Risk — Unknown use of alcohol during pregnancy.
- No Risk — Confirmed absence of prenatal alcohol exposure, which rules out an FAS diagnosis.

Confirmed exposure

Amount, frequency, and timing of prenatal alcohol use can dramatically impact the other three key features of FAS. While consensus exists that alcohol is a teratogen, there is no clear consensus as to what level of exposure is toxic.[23] The CDC guidelines are silent on these elements diagnostically. The IOM and Canadian guidelines explore this further, acknowledging the importance of significant alcohol exposure from regular or heavy episodic alcohol consumption in determining, but offer no standard for

diagnosis. Canadian guidelines discuss this lack of clarity and parenthetically point out that "heavy alcohol use" is defined by the National Institute on Alcohol Abuse and Alcoholism as five or more drinks per episode on five or more days during a 30 day period.[39]

"The 4-Digit Diagnostic Code" ranking system distinguishes between levels of prenatal alcohol exposure as *High Risk* and *Some Risk*. It operationalizes high risk exposure as a blood alcohol concentration (BAC) greater than 100mg/dL delivered at least weekly in early pregnancy. This BAC level is typically reached by a 55kg female drinking six to eight beers in one sitting.[24]

Unknown exposure

For many adopted or adult patients and children in foster care, records or other reliable sources may not be available for review. Reporting alcohol use during pregnancy can also be stigmatizing to birth mothers, especially if alcohol use is ongoing.[11] In these cases, all diagnostic systems use an unknown prenatal alcohol exposure designation. A diagnosis of FAS is still possible with an unknown exposure level if other key features of FASD are present at clinical levels.

Related signs

Other conditions may commonly co-occur with FAS, stemming from prenatal alcohol exposure. However, these conditions are considered Alcohol-Related Birth Defects[23] and not diagnostic criteria for FAS.

- Cardiac — A heart murmur that frequently disappears by one year of age. Ventricular septal defect most commonly seen, followed by an atrial septal defect.
- Skeletal — Joint anomalies including abnormal position and function, altered palmar crease patterns, small distal phalanges, and small fifth fingernails.
- Renal — Horseshoe, aplastic, dysplastic, or hypoplastic kidneys.
- Ocular — Strabismus, optic nerve hypoplasia[40] (which may cause light sensitivity, decreased visual acuity, or involuntary eye movements).
- Occasional abnormalities — Ptosis of the eyelid, microophthalmia, cleft lip with or without a cleft palate, webbed neck, short neck, Tetralogy of Fallot, coarctation of the aorta, Spina bifida, and hydrocephalus.

Prognosis

Primary disabilities

The primary disabilities of FAS are the functional difficulties with which the child is born as a result of CNS damage due to prenatal alcohol exposure.[8] Often, primary disabilities are mistaken as *behavior problems*, but the underlying CNS damage is the originating source of a functional difficulty[41] (rather than a mental health condition, which is considered a secondary disability).

The exact mechanisms for functional problems of primary disabilities are not always fully understood, but animal studies have begun to shed light on some correlates between functional problems and brain structures damaged by prenatal alcohol exposure.[4] Representative examples include:

- Learning impairments are associated with impaired dendrites of the hippocampus[42]
- Impaired motor development and functioning are associated with reduced size of the cerebellum[43]
- Hyperactivity is associated with decreased size of the corpus callosum[44]

Functional difficulties may result from CNS damage in more than one domain, but common functional difficulties by domain include:[41][4][45][46] (This is not an exhaustive list of difficulties.)

- Achievement — Learning disabilities
- Adaptive behavior — Poor impulse control, poor personal boundaries, poor anger management, stubbornness, intrusive behavior, too friendly with strangers, poor daily living skills, developmental delays
- Attention — Attention-Deficit/Hyperactivity Disorder (ADHD), poor attention or concentration, distractible
- Cognition — Mental retardation, confusion under pressure, poor abstract skills, difficulty distinguishing between fantasy and reality, slower cognitive processing
- Executive functioning — Poor judgment, Information-processing disorder, poor at perceiving patterns, poor cause and effect reasoning, inconsistent at linking words to actions, poor generalization ability
- Language — Expressive or receptive language disorders, grasp parts not whole concepts, lack understanding of metaphor, idioms, or sarcasm
- Memory — Poor short-term memory, inconsistent memory and knowledge base
- Motor skills — Poor handwriting, poor fine motor skills, poor gross motor skills, delayed motor skill development (e.g., riding a bicycle at appropriate age)
- Sensory integration and soft neurological problems — Sensory integration (SI) disorders, tactile defensiveness, under-sensitive to stimulation
- Social communication — Intrude into conversations, inability to read nonverbal or social cues, "chatty" but without substance

Secondary disabilities

The secondary disabilities of FAS are those that arise later in life secondary to CNS damage. These disabilities often emerge over time due to a mismatch between the primary disabilities and environmental expectations; secondary disabilities can be ameliorated with early interventions and appropriate supportive services.[8]

Six main secondary disabilities were identified in a University of Washington research study of 473 subjects diagnosed with FAS, PFAS (partial fetal alcohol syndrome), and ARND (alcohol-related neurodevelopmental disorder):[8][4]

- Mental health problems — Diagnosed with ADHD, Clinical Depression, or other mental illness, experienced by over 90% of the subjects
- Disrupted school experience — Suspended or expelled from school or dropped out of school, experienced by 60% of the subjects (age 12 and older)
- Trouble with the law — Charged or convicted with a crime, experienced by 60% of the subjects (age 12 and older)
- Confinement — For inpatient psychiatric care, inpatient chemical dependency care, or incarcerated for a crime, experienced by about 50% of the subjects (age 12 and older)

- Inappropriate sexual behavior — Sexual advances, sexual touching, or promiscuity, experienced by about 50% of the subjects (age 12 and older)
- Alcohol and drug problems — Abuse or dependency, experienced by 35% of the subjects (age 12 and older)

Two additional secondary disabilities exist for adult patients:[8][4]

- Dependent living — Group home, living with family or friends, or some sort of assisted living, experienced by 80% of the subjects (age 21 and older)
- Problems with employment — Required ongoing job training or coaching, could not keep a job, unemployed, experienced by 80% of the subjects (age 21 and older)

Protective factors and strengths

Eight factors were identified in the same study as universal protective factors that reduced the incidence rate of the secondary disabilities:[8][4]

- Living in a stable and nurturant home for over 72% of life
- Being diagnosed with FAS before age six
- Never having experienced violence
- Remaining in each living situation for at least 2.8 years
- Experiencing a "good quality home" (meeting 10 or more defined qualities) from age 8 to 12 years old
- Having been found eligible for developmental disability (DD) services
- Having basic needs met for at least 13% of life
- Having a diagnosis of FAS (rather than another FASD condition)

Malbin (2002) has identified the following areas of interests and talents as strengths that often stand out for those with FASD and should be utilized, like any strength, in treatment planning:[45]

- Music, playing instruments, composing, singing, art, spelling, reading, computers, mechanics, woodworking, skilled vocations (welding, electrician, etc.), writing, poetry

Treatment

There is no cure for FAS, because the CNS damage creates a permanent disability, but treatment is possible. Because CNS damage, symptoms, secondary disabilities, and needs vary widely by individual though, there is no one treatment type that works for everyone. Instead, comprehensive, multi-model approaches based on the needs of the patient must be used. Several treatment models have been identified, but regardless of the predominant approach, most in the current literature recommend multiple types of interventions to ameliorate the negative effects.

Medical interventions

Traditional medical interventions (i.e., psychoactive drugs) are frequently tried on those with FAS because many FAS symptoms are mistaken for or overlap with other disorders, most notably ADHD.[47] For instance, an FAS patient who is inattentive, does not complete schoolwork, and cannot stay seated

has characteristics that an untrained person could easily mistake as ADHD, especially if the patient is not yet diagnosed with FAS. A common course of action would be a medication referral to a pediatrician, who might recommend a trial of Ritalin for the symptoms.

Medications are often important in treating FAS, but should be used in conjunction with other intervention approaches to address the multiple disabilities that arise from FAS.

Behavioral interventions

Traditional behavioral interventions are predicated on learning theory, which is the basis for many parenting and professional strategies and interventions.[45] Along with ordinary parenting styles, such strategies are frequently used by default for treating those with FAS, as the diagnoses Oppositional Defiance Disorder (ODD), Conduct Disorder, Reactive Attachment Disorder (RAD), etc. often overlap with FAS (along with ADHD), and these are sometimes thought to benefit from behavioral interventions. Frequently, a patient's poor academic achievement results in special education services, which also utilizes principles of learning theory, behavior modification, and outcome-based education.

Because the "learning system" of a patient with FAS is damaged, however, behavioral interventions are not always successful, or not successful in the long run, especially because overlapping disorders frequently stem from or are exacerbated by FAS.[45] Kohn (1999) suggests that a rewards-punishment system in general may work somewhat in the short-term but is unsuccessful in the long-term because that approach fails to consider content (i.e., things "worth" learning), community (i.e., safe, cooperative learning environments), and choice (i.e., making choices versus following directions).[48] While these elements are important to consider when working with FAS and have some usefulness in treatment, they are not alone sufficient to promote better outcomes.[45] Kohn's minority challenge to behavioral interventions does illustrate the importance of factors beyond learning theory when trying to promote improved outcomes for FAS, and supports a more multi-model approach that can be found in varying degrees within the advocacy model and neurobehavioral approach.

Developmental framework

Many books and handouts on FAS recommend a developmental approach, based on developmental psychology, even though most do not specify it as such and provide little theoretical background. Optimal human development generally occurs in identifiable stages (e.g., Jean Piaget's theory of cognitive development, Erik Erikson's stages of psychosocial development, John Bowlby's attachment framework, and other developmental stage theories). FAS interferes with normal development,[46] which may cause stages to be delayed, skipped, or immaturely developed. Over time, an unaffected child can negotiate the increasing demands of life by progressing through stages of development normally, but not so for a child with FAS.[46]

By knowing what developmental stages and tasks children follow, treatment and interventions for FAS can be tailored to helping a patient meet developmental tasks and demands successfully.[46] If a patient is delayed in the adaptive behavior domain, for instance, then interventions would be recommended to target specific delays through additional education and practice (e.g., practiced instruction on tying shoelaces), giving reminders, or making accommodations (e.g., using slip-on shoes) to support the desired functioning level. This approach is an advance over behavioral interventions, because it takes the patient's developmental context into account while developing interventions.

Advocacy model

The advocacy model takes the point of view that someone is needed to actively mediate between the environment and the person with FAS.[4] Advocacy activities are conducted by an advocate (for example, a family member, friend, or case manager) and fall into three basic categories. An advocate for FAS: (1) interprets FAS and the disabilities that arise from it and explains it to the environment in which the patient operates, (2) engenders change or accommodation on behalf of the patient, and (3) assists the patient in developing and reaching attainable goals.[4]

The advocacy model is often recommended, for example, when developing an Individualized Education Program (IEP) for the patient's progress at school.[47]

An understanding of the developmental framework would presumably inform and enhance the advocacy model, but advocacy also implies interventions at a systems level as well, such as educating schools, social workers, and so forth on best practices for FAS. However, several organizations devoted to FAS also use the advocacy model at a community practice level as well.[49]

Neurobehavioral approach

The neurobehavioral approach focuses on the neurological underpinnings from which behaviors and cognitive processes arise.[45] It is an integrative perspective that acknowledges and encourages a multi-modal array of treatment interventions that draw from all FAS treatment approaches. The neurobehavioral approach is a serious attempt at shifting single-perspective treatment approaches into a new, coherent paradigm that addresses the complexities of problem behaviors and cognitions emanating from the CNS damage of FAS.

The neurobehavioral approach's main proponent is Diane Malbin, MSW, a recognized speaker and trainer in the FASD field, who first articulated the approach with respect to FASD and characterizes it as "*Trying differently rather than trying harder.*"[50] The idea to *try differently* refers to trying different perspectives and intervention options based on effects of the CNS damage and particular needs of the patient, rather than *trying harder* at implementing behavioral-based interventions that have consistently failed over time to produce improved outcomes for a patient. This approach also encourages more strength-based interventions, which allow a patient to develop positive outcomes by promoting success linked to the patient's strengths and interests.[45]

Public health and policy

Treating FAS at the public health and public policy levels promotes FAS prevention and diversion of public resources to assist those with FAS.[4] It is related to the advocacy model but promoted at a systems level (rather than with the individual or family), such as developing community education and supports, state or province level prevention efforts (e.g., screening for maternal alcohol use during OB/GYN or prenatal medical care visits), or national awareness programs. Several organizations and state agencies in the U.S. are dedicated to this type of intervention.[49]

Prevention

The only certain way to prevent FAS is to simply avoid drinking alcohol during pregnancy.[4] Some studies have shown that light to moderate drinking during pregnancy might not pose a risk to the fetus, although no amount of alcohol during pregnancy can be guaranteed to be absolutely safe.[51][52][53] The Royal College of Obstetricians and Gynaecologists conducted a study of over 400,000 women, all of whom had consumed alcohol during pregnancy. No case of fetal alcohol syndrome occurred and no adverse effects on children were found when consumption was under 8.5 drinks per week.[54] A review of research studies found that fetal alcohol syndrome only occurred among alcoholics; no apparent risk to the child occurred when the pregnant women consumed no more than one drink per day. [55] A study of moderate drinking during pregnancy found no negative effects and the researchers concluded that one drink per day provides a significant margin of safety, although they did not encourage drinking during pregnancy.[56] A study of pregnancies in eight European countries found that consuming no more than one drink per day did not appear to have any effect on fetal growth. A follow-up of children at 18 months of age found that those from women who drank during pregnancy, even two drinks per day, scored higher in several areas of development.[57] An analysis of seven medical research studies involving over 130,000 pregnancies found that consuming two to 14 drinks per week did not increase the risk of giving birth to a child with either malformations or fetal alcohol syndrome.[58]

In the United States, the Surgeon General recommended in 1981, and again in 2005, that women abstain from alcohol use while pregnant or while planning a pregnancy, the latter to avoid damage in the earliest stages of a pregnancy, as the woman may not be aware that she has conceived.[1] In the United States, federal legislation has required that warning labels be placed on all alcoholic beverage containers since 1988 under the Alcoholic Beverage Labeling Act.

See also

- Fetal Alcohol Spectrum Disorder
- Recommended maximum intake of alcoholic beverages
- Alex Burril's Guide to F.A.S.

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External links

- Whitecrow Village FASD Society (http://www.whitecrowvillage.org)
- Fetal alcohol syndrome (http://www.dmoz.org/Health/Reproductive_Health/Pregnancy_and_Birth/Complications/Fetal_Al at the Open Directory Project
- Congressional Caucus on Fetal Alcohol Spectrum Disorders (http://www.house.gov/pallone/fasd_caucus/welcome.shtml)
- Fetal Alcohol Syndrome Diagnostic & Prevention Network (FAS DPN)

(http://depts.washington.edu/fasdpn/)
- CDC's National Center on Birth Defects and Developmental Disabilities (http://www.cdc.gov/ncbddd/fas/default.htm)
- Foetal Alcohol Syndrome Aware UK (http://www.fasaware.co.uk/)
- Iceberg—a quarterly international educational newsletter on FASD (http://www.FASiceberg.org/)
- Fetal Alcohol Syndrome prevention campaign in South Africa (http://www.fasfacts.org.za/)

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